is called for, and to leave it would be comparable to leaving a gangrenous appendix which had been freed from all its adhesions and lay well separated from the abdominal cavity by gauze pads. To drain and leave such a gall-bladder means continued absorption and probably an early death from sepsis. The relief following the removal of an organ in such a condition is well illustrated in my own case by the immediate cessation of symptoms and a fall in the leucocyte count from 37,600 to 12,600 in twenty-four hours. Regarding the technique of the procedure of cholecystectomy in the presence of inflammation of even a milder type than was here present, I am convinced that the advice of Mayo, who states that the cystic duct should not be ligated, is sound, and I am exceedingly glad that in my case the gall-bladder separated and came away before I committed what I now believe would have been an error. There must, in all such cases, be considerable cholangitis present, and nothing gives such relief in this condition, or so quickly arrests it as external drainage.

JAUNDICE, WITH REPORTS OF INTERESTING ILLUSTRATIVE CASES—A CONTRIBUTION TO THE TOXIC FORMS OF THIS CONDITION.*

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Most writers upon the subject of jaundice arrange all cases under two heads—obstructive and non-obstructive, or extrahepatic and intrahepatic jaundice. The data at hand, however, are insufficient to permit of a clear discrimination and an accurate classification in this manner. Furthermore, several possible causes may exist in a given case of jaundice, and it is often difficult to discover which of these is the preponderating or active factor in the condition. In certain cases of obstructive jaundice there is associated a pathological condition of the blood, or of the hepatic cells, one or both. Hence, the great variety and uncertainty of the causes prevent a satisfactory classification at present writing.

There are few diseases of the liver and of the bile passages that are invariably unattended by jaundice. In this group, however, may be placed amyloid liver and fatty liver. On the other hand, there are many hepatic diseases and conditions in the course of which jaundice may rarely occur. Among rare instances may be mentioned cholecystitis, needles, fruit-kernels, distomata, and round worms in the choledoch duct; echinococci, stagnation of bile in the intrahepatic biliary passages, in consequence of interference with the descent of the diaphragm (as in diaphragmatic pleurisy on the right side), so that the propulsion of the bile from the liver to the intestine does not occur (Schuppell); inspissated bile, lipoma, and polypus growing from the wall of the biliary canal, secondary syphilis, and, finally, kinks in the common duct in consequence of displacement of the liver. Frerichs[1] speaks of jaundice caused by an accumulation of pigment in the canaliculi lying between the hepatic cells, as, for example, in malignant malarial fevers; also of an etiological variety occasioned by hemorrhage that drains the vena porta, as in yellow fever. In the latter event there occurs disturbance in the hepatic circulation, with abnormal diffusion of bile and, at times, jaundice. That a paralytic and spastic jaundice occurs, as the older writers contend, is more than doubtful. The commoner causes for both intrahepatic and extrahepatic jaundice are too well known to require enumeration here. Besides, I purpose to consider principally so-called toxic jaundice, giving brief notes of a few illustrative cases. It is sometimes impracticable to distinguish clinically between instances of toxæmic origin and those due to obvious obstruction. Moreover, the fact that, as previously stated, the different forms, etiologically considered, may co-exist is to be recollected.

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In the course of numerous affections that are accompanied by profound toxæmia jaundice may be observed. The toxic form also arises from the action of certain poisons, as ether, chloroform, and the like. The question, Is there a true toxæmic or diffusion icterus? is no longer, as formerly, a vexed one. A number of hypotheses, however, have been advanced to explain its occurrence, with doubtful justifications in most instances. Liebermeister contends that under conditions of pathological change the liver cells cannot retain their secretions.

The theory of Cornil[2] probably explains the mode of origin of many cases at least; it is that the epithelial lining of the smaller bile ducts becomes swollen, and an obstruction is thus produced.

Within the past few years I have met with cases, some of which are reported below, that apparently tend to support in the main the view advanced by Frerichs, and upheld by Murchison,[3] that in many instances of toxic jaundice an excessive amount of bile pigment is secreted, with subsequent reabsorption of the redundant quantity from the intestines. It will be seen later that while the primary and striking pathological feature may be found in the blood, and the amount of bile secreted may be in excess, there is probably in all such cases of jaundice temporary obstruction in the fine biliary canals as the result of increased viscosity of the bile or an intrahepatic cholangitis; hence, reabsorption, not from the intestines, but from the liver, occurs. That we have no means of determining with accuracy the quantity of bile generated per diem, however, is generally conceded, so that this theory lacks demonstrative proof; it rests solely upon clinical testimony. On the subject of polycholia due to ether narcosis, which falls under this head, the literature is peculiarly silent; this is even more true as regards the report of any undoubted cases. In this connection the case of jaundice reported below, following the anæsthetic state, seems worthy of being recorded:

Case I.—Mrs. A. M. S., aged twenty-six years, childless, an actress, was admitted to the gynecological wards of the Medico-Chirurgical Hospital February 27, 1902, under the care of Dr. W. Easterly Ashton.

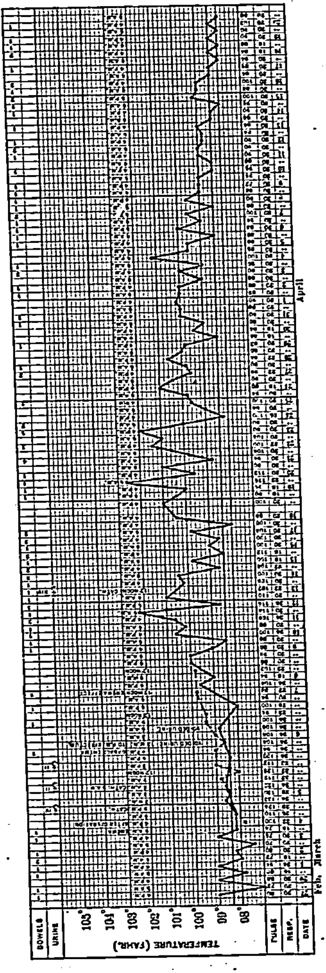
The family history revealed the death of the father from smallpox; also that of the mother from some unknown cause, and of one aunt who died of chronic Bright's disease. The patient had had some of the diseases of childhood; malaria at fourteen, with occasional recurrences since then; peritonitis when sixteen years old, lasting four months; from the age of thirteen until the present she has had tonsillitis at intervals. Since the attack of peritonitis the menstruation has been both profuse and painful. In September, 1901, she suddenly developed acute gastric symptoms, as eructations, with severe pain in the epigastric and the right hypochondriac regions. No history of jaundice appeared that could be elicited now, and the duration of the illness was about one week.

On admission (February 27th) the patient presented the symptoms of bilateral ovarian disease, and on March 4th was operated upon by Dr. Ashton. Cœliotomy revealed a pyosalpinx on one side and a hydropyosalpinx on the other. The case progressed favorably until March 7th (except for persistent vomiting, with retching, in consequence of the etherization), when there appeared slight fever and distress after the feedings; slight pains referred to the epigastrium, right hypochondrium, and the region of the small intestines. Up to this time the bowels had been confined, but now, as the result of a mild saline laxative, diarrhœa set in and soon became a troublesome symptom, there being six to eight movements in the twenty-four hours. The dejecta were of a dark greenish-yellow color in appearance, not offensive, and apparently contained much bile.

On March 12th jaundice appeared, but was slight; the next day the skin and scleræ showed a distinct, moderately deep, saffron hue, not much gastric but more intestinal disturbance, and low irregular fever. (See temperature Chart 1.) The physical signs indicated slight enlargement of the liver; moderate tenderness to palpation over the liver, more marked over the gall-bladder area and intestines. The abdominal wound was reported to be in good condition. Bradycardia absent. The urine analysis showed the presence of bile-pigment, traces of albumin, and a few narrow granular casts. In color it was a dark greenish-yellow, and when shaken displayed a yellow froth.

March 14th. Some pain, which at times radiates to the right shoulder and down the right flank, and tenderness over the region of the gall-bladder, but this organ could not be felt. The jaundice continued, and the urinary examination gave the same result as noted above. The stools still numbered six to eight daily, and were deeply bile-

CHART 1.


BOWELS
URINE
TEMPERATURE (FAHR.)
105°
104°
103°
102°
101°
100°
99°
98°
PULSE
RESP.
DATE
Feb.
March
April

stained. The blood examination gave the following result: Red corpuscles, 3,090,000; hæmoglobin, 60 per cent.; white corpuscles, 27,000. The increase in leucocytes affected principally the polymorphonuclear elements—a simple leucocytosis. A few poikilocytes and nucleated red cells were noted. March 17th, the jaundice and the local symptoms began to disappear.

19th. The skin and conjunctivæ are almost free from discoloration; neither bile, albumin, nor casts were present in the urine, but the bowel movements are still frequent, seven to eight per day; dark greenish-brown in color and, on testing, contained bile pigment.

20th. Jaundice practically absent, patient comfortable except from pain in lower abdominal region. The stools were less frequent—three to four daily, and they contained less bile. About this time, or possibly a couple of days earlier, an abscess in the wound was discovered and properly treated.

April 4th. The patient's general condition greatly improved, the bowels were moving once or twice daily, and the stools presented a normal appearance. An examination of the blood resulted as follows: Red cells, 3,730,000; whites, 20,000; hæmoglobin, 69 per cent. The case is well advanced in convalescence, as the temperature chart also corroborates. At no time during the course of the jaundice was bradycardia present.

The nature of the jaundice in this case is difficult to determine. Was it a case of polychromia or polycholia? In this condition the jaundice results from the dissolution of red blood corpuscles, whereby a large amount of hæmoglobin is set free in the blood current and is converted by the liver cells into bile pigment. The increased formation of bile pigment is invariably attended with increased viscosity of the bile, and the altered, inspissated, hepatic secretion opposes a temporary obstruction to the onward movement of the bile, followed by its absorption. This position is pretty firmly established from the experimental side by the labors of Stadelmann,[4] W. Hunter, Minkowsky and Naunyn.[5] Minkowsky and Naunyn submitted geese and ducks to the action of arseniuretted hydrogen, both before and after extirpation of the liver, and found that while hæmoglobin is present in the urine after removal of the liver, bilirubin is always absent.

Commenting upon the more recent experimental investigations into so-called "hematogenous" jaundice, Lazarus-Barlow[6] states: "It is now generally, though not universally, held (1) that a hematogenous jaundice does not exist; (2) that all varieties of jaundice are hepatogenous and due to reabsorption of bile formed in the liver; (3) the reabsorption is due to the presence of an obstruction in the bile ducts, whether this obstruction is obvious—*e. g.*, a gallstone—or not. Jaundice, therefore, only occurs as the result of blood destruction, when the liver seizes on the blood pigment, converts it into bile pigment, and returns it in bile to the blood." Since neither the polychromia nor the polycholia would occur without the primary blood destruction in these cases, Murchison has suggested that the term "hematogenous" is appropriate in a certain sense, at least.

The evidence before adduced renders it certain that in all cases of jaundice an impediment to the onflow of the bile exists, or, in other words, that the liver is necessary to the production of jaundice. The term "hepatogenous" jaundice may be, therefore, appropriately applied to this symptom in every instance, but it seems to me that it would be advisable to regard as obsolete the term "hematogenous" and to retain that of "toxæmic jaundice" to designate such cases of hepatogenous form as that reported above, as well as all others in which blood destruction is a strong pathological feature. The increased hæmolysis in my case, if it actually existed, was most probably due in great part to the ether. The question, however, arises, Was not the jaundice dependent upon a cholecystitis associated with biliary obstruction? While some of the clinical symptoms and signs bore a close resemblance to cholecystitis, the nature of the dejecta and the brevity of the attack would tend to exclude the latter affection.

Moreover, the local phenomena were scarcely of sufficient intensity to warrant a diagnosis of suppurative cholecystitis; and the physical signs were too indefinite to be of diagnostic value. Again, owing to the slight suppuration in the wound, the presence of leucocytosis and irregular fever, the jaundice might be attributed to a septic process. It is to be recollected, however, that the slight septic symptoms persisted after the jaundice had disappeared. Moreover, in my experience, jaundice of the same degree of intensity has never occurred in so mild a grade of septicæmia. Was the patient affected with a duodenitis that acted as the causative factor? The usual section diet had been taken both prior and subsequent to the operation; it was not thought likely that this had produced duodenal catarrh. The presence of an apparently copious biliary secretion which was discharged per rectum also tends to exclude the possibility of the existence of obstruction in the common duct due to duodenitis. It was most probably true of my case that the abnormally large quantity of bile pigment suddenly generated occasioned an increased viscosity of the biliary secretion and consequent blocking of the hepatic canaliculi. This obstruction, however, was easily removed by a laxative, as compared with that due to catarrhal lesions, hence the comparatively short course of the jaundice. In my case bilious-looking stools promptly followed the administration of a saline laxative three days after operation.

As stated above, bradycardia was not present, while this symptom is the rule in instances of obstructive jaundice. The old view that bradycardia in pure hepatogenous jaundice is due to the action of the complete biliary secretion upon the heart, while in toxæmic jaundice the absence of the bile salts from the blood accounts for a normal pulse rate in the latter variety, is no longer held. Strümpell contends that the infrequency of the pulse is due to the action upon the heart of the taurocholate of sodium. *Per contra,*[6] as pointed out by Lazarus-Barlow, the assertion as to the absence of bile salts in toxic jaundice is not very reliable. Moreover, Stadelmann found them present in the non-obstructive jaundice caused by phosphorus, arseniuretted hydrogen, and other substances; and Naunyn found them in two cases of pyæmia in which the hepatic ducts were free. To account for the difference in respect to the pulse rate between toxic and hepatogenous jaundice is impossible at present writing. The extent of dissolution of the blood in the course of ether narcosis necessary to produce polycholia or polychromia is not known. The condition following the every-day run of operations is exceedingly rare, as shown by an examination of the literature. But that blood destruction occurs during the anæsthetic state has been absolutely proven. Da Costa and Kalteyer[7] have studied the blood in fifty cases, and have concluded that invariably etherization produces increased hæmolysis, following which the rapidly regenerated cells are imperfectly supplied with hæmoglobin. They believe that those who affirm the contrary are possibly misled by the blood concentration that results from the preliminary treatment which is often added to by the sweating during the anæsthetic state. These authors also maintain that the color index always falls and that the number of corpuscles always increases.

Through the kind aid of Dr. L. Napoleon Boston, I can give the results of a few experiments upon rabbits with ether administered by inhalation. The first series of experiments was upon two healthy female rabbits. The blood of each animal was examined directly before administering the ether, and again twenty minutes after the anæsthetic state was reached, at which time the ether was withdrawn. Forty minutes later (one hour after complete anæsthesia was reached) the blood was examined, and later daily until the animal's blood had returned to the normal.

EXPERIMENT I. Female rabbit, pregnant; weight 2 kilos, 130 grammes. Red cells, 4,462,500; white cells, 16,000; hæmoglobin, 60 per cent. Stained specimens presented nothing abnormal. Ether for twenty minutes (℥vj Squibb's). The red cells, 5,750,000; white cells, 24,000; hæmoglobin, 50 per cent. Red cells much distorted and presented decided variation in size, microcytes being numerous. A number of nucleated red cells (mostly normoblasts) were seen; many large polynuclear leucocytes showing irregular basophilic granules were observed.

One hour after ether: Weight 2 kilos, 110 grammes (lost 20 grammes). Red cells, 3,567,600; white cells, 24,000; hæmoglobin, 52 per cent. Poikilocytosis extreme. Microcytes and macrocytes numerous. Many crenated cells were seen in every field. White cells were at last noted.

Twenty-four hours after ether: Hæmoglobin, 30 per cent. Red cells are poor in staining properties, and often stain irregularly, some taking scarcely any of the eosin; poikilocytosis is marked. The polymorphonuclear leucocytes often contain large irregular granules, which are stained brownish by both the eosin and hæmotoxylin. Many cells resembling the "myelocyte" were also observed. Nucleated red cells (normoblasts) were not common.

Forty-eight hours after ether: Red cells, 4,500,000; white cells, 6000; hæmoglobin, 46 per cent. The red and white cells display but moderate variation from the normal. Seventy-two hours after ether, red cells, 4,980,000; white cells, 10,660; hæmoglobin, 47 per cent. One hundred and four hours after ether, hæmoglobin had reached 51 per cent., and on the following day 55 per cent. Otherwise normal.

Experiment II. Female rabbit, weight 3 pounds 3 ounces. Red cells, 3,960,700; white cells, 4200; hæmoglobin, 65 per cent. Twenty minutes after ether (℥iv Squibb's). Red cells, 4,375,000; white cells, 6500; hæmoglobin, 55 per cent. Poikilocytosis was marked, and crenated cells were not uncommon. Microcytes and macrocytes numerous. White cells appeared as normal.

One hour after ether: Weight 3 pounds. Red cells, 3,280,000; white cells, 8500; hæmoglobin, 58 per cent. Poikilocytosis extreme, and especially prominent is the variation in size of reds. Red cells do not stain equally. White cells, polynuclears, display many coarse, brownish granules. Nucleated red cells are not uncommon.

Twenty-four hours after ether: Red cells, 4,690,000; white cells, 6200; hæmoglobin, 43 per cent. Red cells stained irregularly, and many appeared as a mere shadow. Poikilocytosis was but moderate as compared to that observed in Rabbit No. 1. Crenated cells were common, as were also nucleated red cells. Forty-eight hours after ether: Hæmoglobin, 58 per cent. Otherwise as last noted. Sixty hours after ether: Red cells, 4,810,000; white cells 6800; hæmoglobin, 64 per cent. General appearance of stained specimens was as normal.

Experiment III. Female rabbit; weight, 1 kilo, 9845 grammes; was etherized for one hour, Squibb's ether (℥j) employed. Loss in weight 340 grammes. During the first twenty minutes hæmoglobin fell from 82 to 76 per cent., but at the end of an hour it had fallen to 72 per cent. During the following twenty-four hours the reduction in the hæmoglobin was very slight, and at the end of forty-eight hours it had returned to the normal. Leucocytes increased from 6200 to 13,000, while the red cells were, before ether, 6,230,000; twenty minutes after ether, 5,760,000; one hour after ether, 5,430,000; twenty-four hours after ether, 5,210,000; forty-eight hours after ether, 6,310,000; eight days after ether, 6,475,900. The changes in the stained specimens were practically those observed in connection with Experiment II., but were less pronounced.

With a view to determining as precisely as possible the effects of ether administered by inhalation upon the blood, a human subject was next selected, without any special preparation for an anæsthetic.

Experiment IV. J. H., male, aged thirty-three years. Irish extraction, who was apparently in perfect health, except for slight ankylosis of the right knee, resulting from an injury, for which condition he was placed in my ward at the Medico-Chirurgical Hospital. His blood was examined May 1, 1902, which gave hæmoglobin, 86 per cent.; red cells, 4,280,000; white cells, 7600. Stained specimens were found to be normal in every respect. About five hours after a full meal (breakfast) he was given ether, Squibb's, for twenty minutes, at which time the ether was withdrawn and the blood examination repeated; hæmoglobin, 79 per cent.; red cells, 6,150,000; white cells, 16,000. There was no evidence of poikilocytosis; few microcytes were seen; all the erythrocytes stained normally. The increase in polymorphonuclear leucocytes was apparently well-marked. No myelocytes found.

An hour later, hæmoglobin, 74 per cent.; red cells, 4,170,000; white cells, 9800. Many of the red cells were seen to stain feebly with eosin, at times appearing as mere shadows, and there was decided variation in the size of the erythrocytes (microcytes). A differential count of the leucocytes gave polymorphonuclear, 81.91 per cent.; myelocytes, 4.94 per cent.; transitional, 10 per cent.; large mononuclears, 1.11 per cent.; small lymphocytes, 1 per cent.; eosinophiles, 1 per cent.

Twenty-four hours after ether it was reported that the patient had not vomited after the anæsthetic, and that he had taken food and drink twelve hours after the ether was withdrawn. His blood at this time gave hæmoglobin, 67 per cent.; red cells, 4,576,600; white cells, 13,600. Great variation in the size and staining properties of the erythrocytes was observed, and there was pronounced distortion of many of the larger cells. Certain cells stained unevenly, and the general appearance of these cells was not unlike those found in severe grades of anæmia. Leucocytes presented practically the same appearance noted at the previous examination.

Thirty hours after ether: Hæmoglobin, 78 per cent.; red cells, 4,620,000; white cells, 12,000. Forty-eight hours after ether: Hæmoglobin, 80 per cent.; red cells, 5,700,000; white cells, 10,600. At this time there was a decided increase in the number of small lymphocytes, yet the polymorphonuclear cells formed the greater portion of the leucocytes. Few myelocytes were present. The erythrocytes were apparently normal. Seventy-two hours after ether: Hæmoglobin, 86 per cent.; red cells, 4,780,000; white cells, 7400. On one side of the smears three myelocytes were found, otherwise a stained specimen appeared to be normal.

EXPERIMENT V. Animal used for Experiment II. was again etherized May 2, 1902 (seventeen days after the first anæsthesia). Animal now in apparently perfect health. Ether (Squibb's) was given for one hour and forty-five minutes. Blood examinations were made immediately before ether, and after the animal had been anæsthetized for one hour, and for one hour and forty-five minutes, respectively: also twenty-four hours and forty-eight hours after the anæsthetic had been withdrawn.

Before ether: Red cells, 3,016,600; white cells, 8000; hæmoglobin, 50 per cent. Stained specimens appeared as normal. One hour after ether: Red cells, 5,120,000; white cells, 12,000; hæmoglobin, 49 per cent. Slight poikilocytosis. One and three-fourths hours after ether: Red cells, 4,440,000; white cells, 13,600; hæmoglobin, 43 per cent. Total quantity of ether employed, 9 drachms. Red cells show great variation in both size, form, and staining properties of the erythrocytes. Cells staining but feebly are common, and usually these are distorted. Leucocytes appear to be normal as to varieties. Twenty-four hours after ether: Red cells, 3,820,000; white cells, 8000; hæmoglobin, 53 per cent. Crenated cells common, otherwise normal. Subsequent examinations revealed similar findings.

Second etherization of Animal III.

EXPERIMENT VI. Eleven days after first ether. Before ether was given, red cells, 4,730,000; white cells, 8000; hæmoglobin, 79 per cent. Stained specimens appear normal. Ether for twenty minutes, giving all the animal could take, hæmoglobin, 57 per cent.; red cells, 7,880,000; white cells, 8000; died five minutes later. Stained specimens showed great variation in the size of the red cells. All the red cells were extremely pale and poor in hæmoglobin except a few microcytes. Many crenated cells and poikilocytes were found. Microcytes were common and macrocytes numerous. Few nucleated reds (normoblasts) were also observed.

It is of interest in this connection to note, as shown by Experiment V., that during a second etherization, seventeen days after the first, the changes in the blood were less pronounced than at the first; also that this loss of hæmoglobin is restored in from twenty-four to forty-eight hours following the anæsthetic. Again, the duration of the anæsthetic state does not appear to influence materially the blood findings.

These few laboratory experiments show clearly that the fall in the color index in primary anæsthesia reaches its lowest level about twenty-four hours after the anæsthetic state is over; also that one-half of this fall takes place during the first hour after anæsthesia, the other half during the succeeding twenty-three hours. The blood regeneration goes on quite rapidly following the anæsthetic condition, but the hæmoglobin is restored more slowly than the erythrocytes; hence the cells are inadequately supplied with hæmoglobin. At the end of twenty-four hours the color index begins to rise rapidly, and it reaches the normal level after the lapse, approximately, of seventy-two hours in man. A few myelocytes, however, were still present at this time.

The polycythæmia noted does not depend solely upon the preparatory treatment, including purgation, since this was purposely omitted in some of the above cases (Case IV.). The decidedly increased blood production immediately following the administration of the anæsthesia would appear to be a factor in bringing about the polycythæmia according to our experiments. It seems to me that the suddenness with which a rather extensive blood destruction takes place during etherization accounts in a satisfactory manner for the occasional occurrence of polycholia, since there occurs an equally rapid liberation of hæmoglobin which, if acted upon by the hepatic cells, must result in an increased amount of bile pigment. Of course, a mixed etiology may obtain in some cases.

CASE II. *Intense Jaundice in Connection with Lobar Pneumonia.*—C. E. V., aged fifty-three years; married; occupation, clerk; first came under my observation March 4, 1901. The family history showed the ancestors to have been unusually free from hereditary taint, except possibly slight traces of gout and hepatic torpidity in the father. The patient had no recollection of having had any disease of childhood, with the exception of measles. Social habits unexemplary; for many years he had suffered from alcoholic inebriety, and more recently had shown symptoms indicative of the early stage of hepatic cirrhosis. Analogous changes were believed to be present in the kidneys, as suggested by the urinary findings. Moreover, at the age of about forty years he contracted syphilis, and the changes in the liver were ascribed, in part at least, to the syphilitic infection. About three years prior to his death the patient had had pneumonia.

For several months previous to his last (fatal) illness the craving for alcohol seemed to be continuous and resistless; it was accompanied by an unusual degree of exposure to wet and low temperature. The last illness began as an influenza, lasting four days, when pneumonia affecting the lower lobe of the right lung set in without the usual ushering-in rigor and other severe invasion symptoms.

During the preliminary or influenza stage the temperature ranged from 99° F. to 101° F., and with the onset of the pneumonia it rose to 103° F., and finally to 104° F. The temperature curve was of irregularly continued type. The respirations varied in rate from 40 to 60 per minute, and the pulse continued to be regular, of fair volume and force until two days prior to death, when it grew quite rapid, compressible, and arrhythmic. Prune-juice expectoration was present. At the end of the seventh or eighth day of the illness signs of consolidation affecting the left base were also in evidence.

Jaundice of moderate intensity developed on the second day of the pneumonia and gradually deepened until it became intense; this was shortly before the fatal termination occurred. The stools were at no time infrequent, and always contained some bile pigment. During the last four days of life the dejecta numbered from four to six daily, and near the close were passed involuntarily. The urine contained bile pigment in abundance, a small percentage of albumin and tube casts, principally of the granular variety.

With reference to the bile pigment, it may be further stated that it appeared to continue to increase in amount as shown by the color of the urine, reaching its highest limit at the time of death, as was stated respecting the icteric discoloration of the bodily surface and conjunctivæ. Death was preceded by marked nervous disturbances—active delirium—signs of intense motor irritation, as shown by subsultus and coarse tremors. On account of the urinary findings, the nervous phenomena were ascribed in part to uræmia. Delirium tremens did not, as had been feared, carry off the patient.

Autopsy, twenty-four hours after death, by Dr. L. Napoleon Boston: Well-nourished male, apparently fifty years of age; everywhere the skin was jaundiced to an extreme degree. There were also many small cutaneous hemorrhages over the chest and abdomen; the superficial veins of the abdomen were enlarged; there was a small umbilical hernia and slight œdema of the hands, feet, and ankles. The percussion note became dull over the right lung at the third interspace, extending downward to a point two inches below the costal margin. The left lung showed slight impairment of the percussion note at the base; only a small incision into the abdomen was allowed. Abdominal fat three inches in thickness; abdominal cavity contained some fluid (removed by undertaker). Viscera including the liver were jaundiced; the stomach showed slight congestion, probably post-mortem.

Duodenum showed no evidence of congestion at any point. The mouth of the ductus communis was in no way obstructed. Upon pressure over the gall-bladder bile escaped into the duodenum. The gall-bladder and ducts presented nothing abnormal on macroscopic examination. The edges of the liver were nodular, and at many points there were decided contractures—attempts at the formation of additional lobes. On the superior surface, near the junction of the right and left lobes, it was decidedly nodular (hobnail). Each of the small liver lobules stood out clearly and was surrounded by a rather broad band of yellowish tissue, which was depressed. The base of the right lung was consolidated, as was also a small portion of the left lung posteriorly. At several places the organ had been entered by the needle and injected with the embalming fluid. The kidneys were not examined for the reason that it was not permitted.

The report of the microscopic findings in the liver by Prof. Coplin was as follows:

The changes were those typical of fatty cirrhosis, with marked cholangitis affecting the intrahepatic excretory ducts. Extensive desquamation of biliary epithelium was observed, and the presence in the smaller bile ducts of bacteria resembling the colon bacilli.

Such cases as the above prove with complete certainty that in some instances of pneumonic jaundice, at least in which there is no obvious macroscopic obstruction to account for the symptom, the microscopic examination may reveal the changes of a cholangitis. J. E. Graham[8] affirms that the jaundice of cardiac insufficiency and of pneumonia is accounted for by the fact that the bile is secreted under low pressure, so that the changes in the circulation may cause it to flow from the cells into the veins rather than into the biliary radicals. Is it not a fact, however, that bile is ordinarily secreted under low pressure? According to certain observers it is of toxic origin, while still others believe it to be due to catarrhal lesions affecting the duodenum and larger excretory ducts of the liver. In the case reported above, the intrahepatic cholangitis was doubtless the active causative factor. May not the increased amount of bile pigment have set up an inflammation of the fine biliary ducts? The obstruction to the outflow could not have been complete, owing to the constant presence of biliary coloring matter in the dejecta. Hence, the toxic element may also have played no inconspicuous part in the causation of the jaundice. The *modus operandi* would be similar to that pointed out in connection with Case I. I have seen two additional cases in which the lesions of an ordinary catarrhal jaundice existed, hence the icteric tint may at times be solely dependent upon the latter changes.

Mallory[9] and Longridge[10] have called attention to foci of necrosis, which they claim are common to the acute infections, including lobar pneumonia. It is of two varieties, central or diffuse, and focal. In the former (central) the lesion begins in the centre of the lobules and spreads peripherally. Focal necrosis may rarely be due to the toxin of pneumonia. Neither form of necrosis, however, was found on histological examination of the liver from my case.

In 1898, Türck[11] showed that when the pneumonic infection is severe, or the patient has low vitality, nucleated red cells, principally normoblasts, and in a lesser degree also megaloblasts appear in the peripheral blood. Maragliano has observed "degenerative" changes in the red cells in severe cases. It is interesting to note that in the observations upon the effect of the anæsthetic (ether) on the blood of our rabbits, an increased number of nucleated red cells, mostly normoblasts, and other degenerative changes were observed. The point is that increased hæmolysis from any cause may induce similar blood changes, and may also be the cause, with the aid of the liver function, of a symptomatic jaundice, as before explained. Again, when the liver is the seat of chronic pathological change, as in the case just reported, the obstruction to the outflow of inspissated and abnormally viscid bile is far greater than in a previously normal organ.

In this connection I desire to direct attention to the question of the causes of death in this and similar cases. It is doubtless true of obstructive jaundice that death is, in the majority of cases, due to the underlying condition that induces the jaundice rather than to the icterus itself. In some cases, however, and more especially those in which an excess of bile pigment is generated, the poisonous effects of the latter may prove fatal. Bouchard regards the biliverdin as the toxic constituent. As clinicians we are not so much concerned with the question of the special ingredient of the biliary secretion that possesses the poisonous properties—a moot point—as with the fact that a serious and even fatal condition (so-called cholæmia) may be suddenly induced by this secretion.

Just how death is brought about from the poisonous action of the bile is imperfectly understood. Much depends upon the previous condition of the kidneys. Moreover, I am firmly of the opinion, based on personal observation, that an acute nephritis is often set up by the bile pigment in the process of elimination through a normal urinary tract. This fact is corroborated by Case V. of my present series (*vide infra*).

It is rare, indeed, to meet with jaundice during the secondary stage of syphilis. Through the kindness of my friend and assistant, Dr. Boston, I am able, however, to present brief notes of an illustrative case:

CASE III.—M. C. D., aged nineteen years; American; prostitute; first came under observation June 10, 1897. Parents and four brothers living and fairly healthy; all, however, are alcoholics.

Past History. She had some of the affections of childhood, afterward continued in good health until six or eight weeks ago, when she developed an eruption, the remains of which show it to have belonged to secondary syphilis. The patient has no knowledge of an initial sore. Three weeks ago she noticed a swelling of the feet and abdomen, at the same time developing a yellow vaginal discharge. A pelvic examination showed a cervical discharge and a cervix that was ulcerated and bleeding. The conjunctivæ and skin were markedly jaundiced, and the feet swollen to twice their normal size. The heart sounds were weak, valvular, and distant, but fairly regular; the liver was enlarged and tender to pressure; otherwise the physical signs were negative.

June 22, 1897. There is a wave of fluctuation over the abdomen; temperature, 101°; pulse, 120; respiration, 24. The patient complained of pain in the region of the liver and at angle of scapula; abdomen much distended.

23*d.* The condition as noted yesterday, except temperature, now 102°, and respiration 30 and difficult; she sits pillowed up in bed; pain severe, and size of abdomen increasing. Lips and tongue fissured, the former bleeding; also oozing of blood from the nose and gums; slight systolic murmur at apex; vomited twice during the day; face distinctly puffy; intense jaundice.

Examination of Urine. Specimen porter color, specific gravity 1020; sediment heavy; reaction acid. Chemically it was found to contain considerable albumin and bile pigment, but no sugar. The microscope revealed bile-stained pus, tube casts (granular and epithelial), and epithelium (vaginal and vesical).

June 24*th.* On the following day the patient greatly improved both locally and generally, although vomits a dark coffee-colored material every few hours.

26*th.* Jaundice still marked; stomach non-retentive, all food and dark-colored fluid vomited; an examination of stools showed bile pigment, epithelium, etc., but no pus.

27*th.* Pain persists in abdomen, but there is no vomiting, and the jaundice is disappearing. At no time was there an absence of bile pigment from the stools. The patient now improved day by day under anti-syphilitic treatment, until at the end of four to five weeks all symptoms had vanished.

The cause of the jaundice in this instance was believed by Dr. E. P. Martin, who saw the patient by request, to be perihepatitis. According to White and Martin,[13] jaundice developing during the secondary period is rarely due to syphilis. The great majority of such cases, when unattended by hepatic enlargement, as in the case herewith reported, are caused by intercurrent affections, such as a catarrhal condition of the bile ducts, "and are neither directly nor indirectly dependent upon constitutional syphilis." It is not likely that a gumma in the portal fissure was the cause of the jaundice in this case, as it had not progressed beyond the second stage. While the jaundice in this case was ascribed to the perihepatitis, although no blood examination was made, it is to be recollected that the syphilitic virus may cause extensive dissolution of the erythrocytes and marked oligochromæmia in the second stage of the affection. In this stage all forms of nucleated reds and poikilocytosis have also been commonly met with, especially when, as in this instance, the patients have belonged to the semistarved, pauper element of society. The blood changes are identical with those characterizing toxæmic jaundice, and they may sustain an indirect causative relation to syphilitic icterus.

The question as to the cause of the fever in this case is an interesting one. Was it occasioned by acute nephritis? or by an intercurrent cholecystitis? or, finally, was it an instance of syphilitic fever? According to the results of the therapeutic tests, it was almost certainly ascribable to syphilis. Janeway[13] and others have reported cases in which fever attended the late manifestations of syphilis. It also occurs in the second stage. I have recently met with a case in a male, aged twenty-five years; it lasted about two weeks, and on several occasions the temperature rose to 102° F. Basset-Smith[14] reports a case in which a mis-diagnosis of malaria had been made on account of an intermittent fever curve. As the nephritic symptoms inducing dropsy disappeared under syphilitic treatment, they were doubtless of syphilitic origin. Allaria[15] also invites attention to early (secondary) syphilitic nephritis, with severe albuminuria, anasarca, and recovery under specific treatment.

Case IV. *Jaundice due to an intercurrent acute inflammatory condition in the course of hepatic cirrhosis.*—T. O. D., aged forty-four years; occupation plumber; single; admitted to the male medical wards of the Medico-Chirurgical Hospital, March 20, 1902. The family history is perfectly good, except that one sister died at the age of twenty-three years of some unknown cause. The patient had had none of the affections of childhood. Two attacks of acute articular rheumatism, the first at thirty years of age, lasting fourteen weeks, and the second at forty-one years, duration five weeks. During convalescence from the last attack of rheumatism, he drank heavily and steadily for a year or more, when he developed evidence of chronic nephritis, including dropsy of the feet and legs. He then abandoned alcohol, was placed upon appropriate treatment, and apparently recovered.

Shortly thereafter, or ten months ago, he had an attack of pneumonia, for which he was treated in the Medico-Chirurgical Hospital. Following this illness he again became intemperate, using whiskey in excess; this he continued until date of admission. The illness that led him to apply for relief began six weeks previously, with indefinite symptoms, as fatigue, anorexia, constipation, and other prodromata of liver cirrhosis.

On March 19th (one day prior to admission) he developed intense pain in the region of the gall-bladder, radiating to the right shoulder, abdomen, and back. Fever developed; it was of irregularly continued type, and did not range high, the maximum temperature not exceeding 105.3° F. There were no rigors; the pulse was moderately accelerated (not slowed). The urine analysis at this time showed the presence of bile pigment, a small percentage of albumin and fine granular and epithelial casts. A physical examination indicated great enlargement of the liver, the lower border reaching to a line one inch below the umbilicus. The gall-bladder area was tender to the touch, but this viscus was not palpable. The second urine analysis, made March 25th, yielded results similar to those of the first, although the albumin was increased in amount to about 1 per cent. The skin was now a deep saffron hue. Fever more marked; patient quite delirious at night, exhibits noisy respiration, and is profoundly prostrated.

March 26th. All symptoms, including the jaundice rapidly abating; the liver is also diminishing in size. Albumoses searched for, but not found at any of the urinary examinations. The stools were not clay-colored, indicating no obstruction in the excretory ducts. This remission in the symptoms lasted a few days and then they reappeared, although less marked. (See Chart 2.)

31st. The jaundice returned with the recurrence of the fever.

April 17th. The temperature has been ranging near to the normal grade for one week, and the jaundice almost entirely vanished.

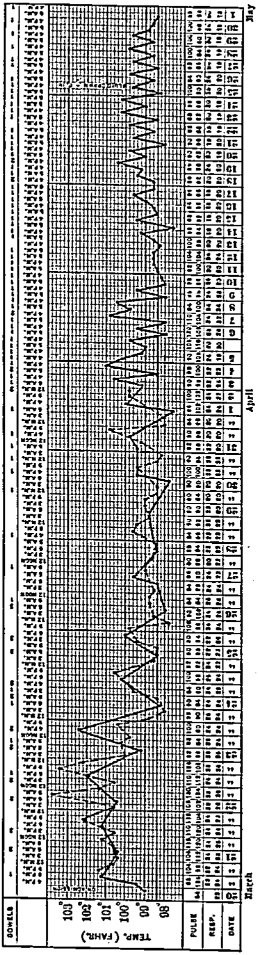
May 1st. The temperature has become nearly normal after a slight rise about ten days ago, and the patient has been growing stronger. Ascites has developed acutely during the past month; abdomen was tapped this day and 108 ounces of bile-stained fluid removed. A subsequent physical examination indicated only moderate hepatic enlargement, the organ having rapidly diminished in size during the previous two weeks. Very slight jaundice remained.

9th. Slowly improving. A blood examination resulted as follows: Reds, 3,790,000; whites, 9000; hæmoglobin, 55. Cells of normal size and form.

That cirrhosis may often be due to multiple causes is universally conceded. Again, many cases have as their principal etiological agent an infecting microbe. Thus tuberculosis, syphilis, malaria, one and all, may cause it. It is more particularly in the hypertrophic form of the disease that the etiology is often obscure. The case I have here reported is to be classed as alcoholic cirrhosis, according to the clear history. It is not unlikely, however, that as regards many cases of this variety of atrophic cirrhosis, our notions concerning etiology may be upset by future investigations.

Adami[16] affirms that in cattle the infective disease known as "Pictou disease" presents hepatic changes similar to those found in cirrhosis in man. The irritant in this case is believed to be a bacteriological toxin,

CHART 2.


BOWELS
TEMP. (FAHR.)
103°
102°
101°
100°
99°
98°
PULSE
RESP.
DATE
March
April
May

which acts as a protoplasmic poison. Hektoen[17] reports a case of cirrhosis of the liver in animals, produced by a member of the colon group, and another that was caused by inoculation of the bacillus which belongs to the pseudodiphtheria group. Cultures were made from the bile-stained peritoneal fluid withdrawn in my case, with the following result: Bouillon cultures at twenty-four hours showed an abundant growth of motile bacilli, which on staining and further study were found to be members of the colon group. No other organisms were present in cultures. Slides were smeared with the peritoneal fluid, and when stained were found to show few bacilli which decolorized by Gram's method. No tubercle bacilli were found. The fluid was lemon-yellow in color, turbid, and presented a flocculent sediment; its specific gravity was 1010, reaction slightly alkaline, and contained about 0.12 per cent. of albumin.

This case also represents a group or class of cases that are by no means uncommon, and in which chronic hepatic disease, most commonly cirrhosis, is suddenly complicated with an acute inflammatory condition. In Case IV., just related, the intercurrent affection was regarded as being cholecystitis.

The principal features in this instance were similar to those of Weil's disease, namely, jaundice, fever, and albuminuria with casts. Rigors were absent, however, and the temperature curve closely resembled that of relapsing fever, as may be seen from a glance at the accompanying fever chart. (See Chart 2.) The intensity of the jaundice also corresponded pretty closely with the degree of elevation of temperature. This so-called relapsing jaundice must, it seems to me, be due to an infection and re-infection, but whether or not the colon bacillus is to be incriminated is still uncertain. Karlinski and others have described an epidemic jaundice of unknown etiology, in which the febrile movement pursued a similar course. These facts tend to prove the infectious nature of the condition, but it may be of varied etiology. H. Neuman[18] goes so far as to state that there is no demonstrable difference between epidemic infectious jaundice and simple catarrhal jaundice, which, he holds, is also due to an infection.

CASE V. *Mixed cirrhosis, biliary and atrophic variety.*—This patient was under the charge of Dr. I. Newton Snively, to whom I am indebted for the notes of the case.

Mr. H. F., aged twenty-eight years; American; weight about 240 pounds; height 5 feet 10 inches; book-keeper by occupation. The family history is good, except that the father had been afflicted many years with epilepsy, and died from apoplexy.

Previous History. He had the ordinary diseases of childhood; measles, mumps, chicken-pox, and whooping-cough. As a boy he complained unusually of shortness of breath upon exertion, such as running and jumping, etc. He has been drinking quite heavily since he was nineteen years of age, and for two years and a half past has taken whiskey in enormous quantities, perhaps from a quart to a quart and a half every twenty-four hours; yet was never intoxicated so as to be unable to attend to his duties as book-keeper, etc.

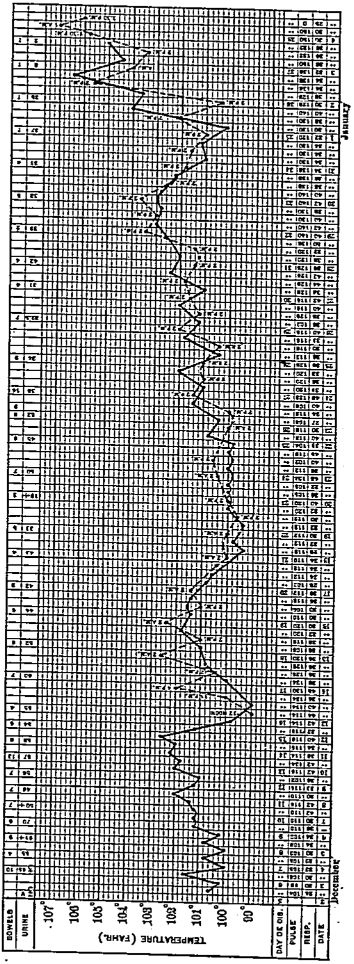
About July, 1900, he noticed a sore on the right upper thigh about two inches back of the scrotum; he attributed it to chafing, but as it refused to heal, he consulted Dr. Clifford Scott, who suspected specific infection, treated him accordingly, and in three or four weeks the sore healed. The patient was an inveterate smoker of cigarettes. Since twenty-one years of age he has been growing stouter, and for two years past has been extremely corpulent—in fact, almost helpless at times. One year before the onset of his present sickness he noticed a dropsical condition of the lower limbs, which later involved the scrotum, and he suffered greatly with shortness of breath, especially upon exertion. He was obliged to sleep propped up with head-rest and pillows for several months. His appetite, however, was enormous, and he ate largely of all kinds of food until the beginning of the present sickness.

The above symptoms, œdema of the lower limbs and scrotum, shortness of breath on exertion, etc., grew gradually worse until on November 29, 1901, he was compelled to stay in his room. At this time the abdomen was enormously enlarged, the limbs and scrotum were swollen to their utmost capacity and were oozing fluid. His pulse and respirations were rapid, as shown by the chart. He was tapped in the abdomen and scrotum a number of times; also the limbs.

Physical exploration, after paracentesis, by palpation and percussion showed an enormously enlarged liver. I saw the patient several times in consultation with Dr. Snively at intervals of several days. During the last month of life the temperature ranged from 99° to 102.3° F., ending with hyperpyrexia (see Chart 3); this was accompanied by delirium, prostration, and other evidences of an acute infection, without our being able to recognize the nature of the morbid process. The skin and conjunctivæ had been jaundiced for about three weeks before death. The tappings show that up to December 7, 1901, 384 ounces of fluid had been drawn off; also estimated 64 ounces on December 8th, which simply represented the oozing from scrotal openings made by the trocar and the oozings from the legs where the trocar had been used. The charts show several later tappings. Urinary analysis showed no sugar, albumin, or casts, except during the last four or five days of his life, when the urinary findings characterizing acute nephritis developed. He died January 4, 1902.

Post-mortem findings twenty-four hours after death by Dr. L. Napoleon Boston: Body of a male, large in stature, face swollen, and skin and conjunctivæ jaundiced. The veins over the abdomen and chest were dilated. A dull percussion note on the right side, commencing three inches above nipple and extending to crest of ilium. On opening the chest 100 c.c. of bloody fluid was found in each pleural sac; right lung pushed up to third rib and the arch of the diaphragm lifted to the same point in nipple line. Hypostatic congestion rather extensive in the left lung, and confined to the base of right lung. The myocardium was contracted, its walls thin, and showed fatty changes on section; the chambers were occupied by dense fibrous clots. Microscopic study of the heart muscle impossible, owing to the embalming fluid. The stomach was normal except that its walls were slightly congested,

CHART 3.


BOWELS
URINE
TEMPERATURE (FAHR.)
107°
106°
105°
104°
103°
102°
101°
100°
99°
DAY OF DIS.
PULSE
RESP.
DATE
December
January

but the ductus communis choledochus was patulous, and a needle could be passed directly into the gall-blader. The gall-bladder was normal and contained about one-half ounce of bile.

Liver. Weight 6040 grammes; color a greenish-yellow, surface smooth, slightly mottled, capsule free, and edges rounded. On section, resistance to the knife was increased; the surface presented a rather granular aspect; the acini were uniformly prominent, elevated, and each surrounded by a band of yellowish-white tissue, which showed slight depression. The needle of the embalming trocar had pierced the organ at several points, hence attempts at section for microscopic study were not altogether satisfactory, although the appearances were in the main those of fatty cirrhosis of the organ. The liver tissue was everywhere clearly and distinctly bile-stained. The kidneys presented the usual naked-eye appearance of acute nephritis, and this was also shown by microscopic study.

A microscopic examination of the liver by Dr. Coplin revealed the following:

"There is a large amount of elastica, which is intralobular and interlobular; enters lobules and often surrounds columns of cells, and at points is increased around smaller bile ducts, and is still more increased around the larger biliary branches. Newly-formed tissue between lobules, which is, for the most part, young and fully formed; fibrous tissue is not so abundant as the gross specimen would have led one to believe. The liver cells show fragmentation of protoplasm and nuclei, and advanced granular degeneration is evident in many cells. Young cell deposits are present, even within the lobules. Uniform fatty infiltration is fairly evenly distributed throughout sections.

"Epithelium of bile ducts is advancedly granular, often fragmented, and often desquamating. Evidence (distinct) of new bile duct formation is wanting. Many of the lobules show intense bile-staining, more pronounced at periphery than at centres. The lobules showing less fatty infiltration, least fragmentation of cells, and least young cell invasion and increase of elastica, are most stained with bile (and are probably secreting bile). Many stained cells are shrunken. Mixed cirrhosis, biliary, and fatty alcoholic varieties."

The features of greatest clinical interest in this case are the intense jaundice, the fever, and the acute nephritis coming on in the course of cirrhosis. A glance at the temperature chart will show that hyperpyrexia, with tendency to daily increase of fever, supervened two days before the occurrence of the fatal termination. The curve also shows a disposition, though incomplete, to relapsing febrile jaundice. Bradycardia was not present at any time; on the other hand, the pulse was accelerated throughout, and during the last three weeks of life much increased in frequency. The post-mortem failed to reveal any localized inflammatory or other lesions, save those of the liver and kidneys, to account for the fever, and it should be recalled that the urinary findings were negative up to within a week of the fatal termination. It seems to have been an instance of febrile jaundice arising in the course of hepatic cirrhosis with the secondary development of acute nephritis. The case bears out the view previously expressed, namely, that febrile, infectious jaundice may be complicated with nephritis, and the latter condition may result from irritation of the renal tissue by the bile pigment in the process of elimination from the system. It is to be borne in mind, however, that in many of the acute infections nephritis due to the local irritant effect of the special toxin is not very uncommon. Uræmic symptoms attended near the close in Case V.

In conclusion, while the cases reported above do not furnish a basis for definite, conclusive inferences, they suggest a few queries and propositions:

1. Is there a true polycholia due to the conversion into bile pigment of the hæmoglobin that is suddenly liberated by certain poisons, as ether, chloroform, and the like, or the toxins of the acute infections, as, for example, lobar pneumonia?

2. Doubtless other causative agents are often operative in cases that fall under 1.

3. As there could be no jaundice without the presence of the liver, the term "hematogenous" is misleading, and should be regarded as obsolete; the term "toxæmic jaundice" may be, however, appropriately applied to the cases of "hepatogenous" icterus, in which the blood changes are a more or less striking pathological feature.

4. May not the jaundice rarely seen in secondary syphilis be toxic in nature, at times at least, rather than purely "hepatogenous?"

5. Admitting that there are well-established causes for the hepatic cirrhosis, more particularly of the atrophic form, the etiology is but imperfectly understood.

6. Clear evidence to show that the hepatic cirrhoses are, in many cases at least, of microbic origin is accumulating.

7. The intercurrent acute inflammatory conditions with jaundice in the course of cirrhosis of the liver are doubtless micro-organismal in nature. (See Cases II., IV., V.)

8. Relapsing febrile jaundice is also to be classified as an infection; it will probably be shown on further investigation to be due to a member of the colon group. (See Case IV.)

9. It is probable that the jaundice of hepatic cirrhosis is, in a certain sense at least, toxic in nature.

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ABSCESSES OF THE LIVER FOLLOWING TYPHOID FEVER.

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JOHN H., aged thirty-four years, came to me June 24, 1901, complaining of pain in the right side; chilly feelings and sweats, and great weakness. He said that three days before he saw me he had left his bed, where he had been confined for six weeks suffering from a disease diagnosed by his attending physician as typhoid fever. The history of this illness was typical for an ordinary case of typhoid fever, except that he felt that he was regaining strength very slowly, and that he had felt a heaviness and dull pain in the right side during the last week that he remained in bed.

The patient's previous history was negative. He suffered from measles when a child, and had smallpox when twenty years of age, but had had no other illness.

The examination showed a well-developed, poorly-nourished and emaciated middle-aged man. His skin seemed somewhat darker than normal, but he was not jaundiced. The mucous membrane of the lips and conjunctivæ was pale. There was marked tenderness in the region of the liver, and I inferred from the physical findings that the liver was diffusely enlarged. Its lower border was well defined and extended a hand's breadth below the costal arch at the outer border of the right rectus muscle. The upper border of relative hepatic dulness extended, in the right mammillary line, to the upper border of the third rib. Examination of the chest showed diminished expansion and respiratory excursion on the right side. The apex of the left lung seemed duller than normal. Harsh breathing was found in the apex of the left lung and over the lower portion of the right. A few moist râles were present in both lungs posteriorly. No friction rubs were heard. The heart was slightly enlarged transversely, and a systolic murmur was present over the pulmonic area. No thrills were found. The pulse was beating 108 times per minute; was not strong, but was regular. The temperature was 101° F. The spleen was not felt. There was no tenderness in the lower abdominal region; and no evidences of fluid in the peritoneal cavity could be found. The remainder of the examination was negative. The urine showed a trace of albumin, but was otherwise normal.

A diagnosis was not made at this time. A diagnosis of cardiac insufficiency, with passive congestion of the liver, was not warranted.